# Clinical review: use of venous oxygen saturations as a goal - a yet unfinished puzzle

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REVIEW

# Clinical review: use of venous oxygen saturations as a goal – a yet unfinished puzzle

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## Abstract

Shock is defined as global tissue hypoxia secondary to an imbalance between systemic oxygen delivery and oxygen demand. Venous oxygen saturations represent this relationship between oxygen delivery and oxygen demand and can therefore be used as an additional parameter to detect an impaired cardiorespiratory reserve. Before appropriate use of venous oxygen saturations, however, one should be aware of the physiology. Although venous oxygen saturation has been the subject of research for many years, increasing interest arose especially in the past decade for its use as a therapeutic goal in critically ill patients and during the perioperative period. Also, there has been debate on differences between mixed and central venous oxygen saturation and their interchangeability. Both mixed and central venous oxygen saturation are clinically useful but both variables should be used with insightful knowledge and caution. In general, low values warn the clinician about cardiocirculatory or metabolic impairment and should urge further diagnostics and appropriate action, whereas normal or high values do not rule out persistent tissue hypoxia. The use of venous oxygen saturations seems especially useful in the early phase of disease or injury. Whether venous oxygen saturations should be measured continuously remains unclear. Especially, continuous measurement of central venous oxygen saturation as part of the treatment protocol has been shown a valuable strategy in the emergency department and in cardiac surgery. In clinical practice, venous oxygen saturations should always be used in combination with vital signs and other relevant endpoints.

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## Introduction

Shock is defined as global tissue hypoxia secondary to an imbalance between systemic oxygen delivery ($DO_2$) and systemic oxygen demand ($VO_2$). Unrecognised and untreated global tissue hypoxia increases morbidity and mortality. Accurate detection of global tissue hypoxia is therefore of vital importance. Physical findings, vital signs, measuring central venous pressure and urinary output are important but insufficient for accurate detection of global tissue hypoxia [1-3]. Measurement of mixed venous oxygen saturation ($SvO_2$) from the pulmonary artery has been advocated as an indirect index of tissue oxygenation [4]. As a result of an extensive debate in the literature [5-7], however, use of the pulmonary artery catheter has become somewhat unpopular. In contrast, insertion of a central venous catheter in the superior vena cava via the jugular of the subclavian vein is considered standard care in critically ill patients. Just like $SvO_2$, the measurement of central venous oxygen saturation ($ScvO_2$) has been advocated in order to detect global tissue hypoxia.

Venous oxygen saturations have been the subject of research for over 50 years, but especially over the past decade the amount of literature describing changes in $ScvO_2$ and $SvO_2$ in critically ill patients, including high-risk surgical patients, increased substantially. This led to high expectations with respect to the use of venous oxygen saturation as a therapeutic goal. The aim of the present review is to summarise the evidence and to discuss the clinical utility of both $SvO_2$ and $ScvO_2$ in the treatment of critically ill patients, including high-risk surgical patients.

We performed a search of the PUBMED database from 1980 to 2010 using combinations of the following terms: $SvO_2$, $ScvO_2$, venous oxygen saturation, venous saturation, critically ill, shock, septic shock, high risk surgery, surgery, operation. The articles published in English were included when published in a peer-reviewed journal. The clinical investigations had to concern adults. Additionally, bibliographies of relevant articles were also screened.

## Physiology

Understanding the physiology of venous saturations is essential for effective application in critically ill patients and during the perioperative period.

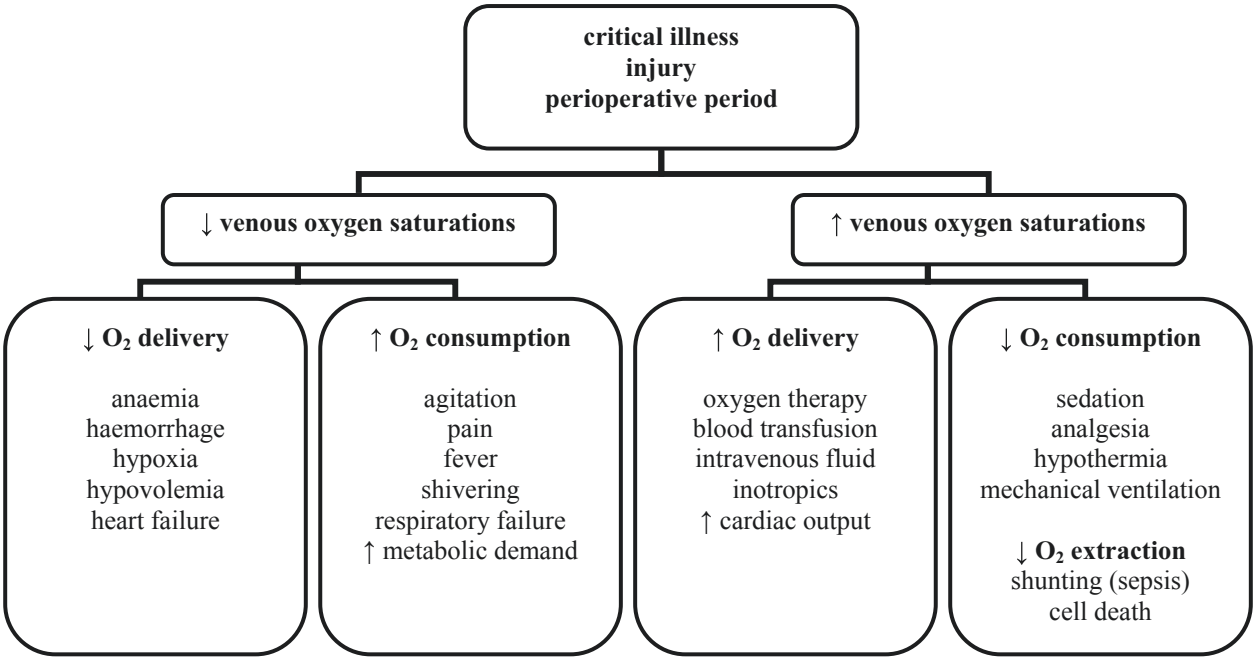


**Figure 1. Multiple physiologic, pathologic and therapeutic factors may influence the value of central venous oxygen saturation.**

$SvO_2$ depends on arterial oxygen saturation ($SaO_2$), the balance between $VO_2$ and cardiac output (CO), and haemoglobin (Hb) levels. According to the Fick principle [8], $SvO_2$ can be described by the following formula:

$$SvO_2 = [SaO_2 - VO_2 / CO] [1 / Hb \times 1.34]$$

Increased $VO_2$ will be compensated by increased CO. If this is not adequate – that is, if oxygen demand is not met – elevated oxygen extraction occurs in the peripheral tissues and consequently $SvO_2$ will drop. $SvO_2$ thus reflects the balance between oxygen delivery and oxygen demand [9]. The normal range for $SvO_2$ is 65 to 75% [4,10]. Low $SvO_2$ is predictive of bad outcome [4,11], whereas normal or supranormal $SvO_2$ (or $ScvO_2$) values do not guarantee adequate tissue oxygenation [12,13]. If tissue is not capable of extracting oxygen (for example, in the case of shunting and cell death), venous return may have a high oxygen content despite persistent cellular hypoxia.

A variety of physiological and pathological changes may influence venous saturation (Figure 1) and thus require different therapeutic interventions. Recognition of the aetiology of any derangement is obligatory for the safe use of venous saturation as a therapeutic goal.

## Central versus mixed venous oxygen saturation

In general there has been considerable debate on equality or interchangeability of $ScvO_2$ and $SvO_2$ [14-16] (see Table 1). In critically ill patients, substituting $SvO_2$ by $ScvO_2$ results in large variability [16-21]. This could in part be explained by modifications of blood flow distribution and oxygen extraction by brain and splanchnic tissue. In this situation, $ScvO_2$ may provide the false impression of adequate body perfusion. Also, whether a positive $ScvO_2$–$SvO_2$ gradient can be used as a marker of greater oxygen utilisation and a predictor of survival remains a subject of debate [20,22,23].

In contrast, other studies have stated that $ScvO_2$ could indeed be used as a substitute for $SvO_2$ [24-26]. For example, Reinhart and colleagues performed continuous measurements of venous oxygen saturations in anaesthetised dogs over a wide range of haemodynamic conditions, including hypoxia, haemorrhage and resuscitation, and described close tracking between $ScvO_2$ and $SvO_2$ [24]. However, correlation was lowest during hypoxia, one of the areas of greatest clinical interest. Nevertheless, precise determination of absolute values for $SvO_2$ from $ScvO_2$ was not possible, as was seen before [21,27-29].

Additionally, the relationship between CO or the cardiac index and venous saturations has been evaluated in critically ill patients. So far, the results have been inconclusive for both $SvO_2$ and $ScvO_2$. Larger trials are needed before clinical recommendations can be made regarding their clinical use [19,30-33].

## Clinical use of venous oxygen saturations

### Cardiac failure

Venous oxygen saturations have been shown to have diagnostic, prognostic, and therapeutic qualities in critically ill patients with acute myocardial infarction (see

**Table 1. Studies comparing mixed venous oxygen saturation and central venous oxygen saturation**

| Study | Design and subjects | Results | Conclusions |
|---|---|---|---|
| Varpula and colleagues [14] | *n* = 16; septic shock; ICU; 72 paired samples | Mean $SvO_2$ below mean $ScvO_2$ at all time points; bias of difference 4.2%<br>95% limits of agreement –8.1 to 16.5%; difference correlated with CI and $DO_2$ | Difference between $ScvO_2$ and $SvO_2$ varied highly; $SvO_2$ cannot be estimated on basis of $ScvO_2$ |
| Martin and colleagues [16] | *n* = 7; 580 comparative measurements; critically ill patients; ICU; with and without interventions | Difference ≥5% in 49% during periods of stability and in 50% during periods with therapeutic interventions | $ScvO_2$ monitoring not reliable |
| Chawla and colleagues [17] | *n* = 32 postsurgical and *n* = 21 medical; ICU | $SvO_2$ consistently lower than $ScvO_2$ with mean (± SD) bias –5.2 ± 5.1% | $SvO_2$ and $ScvO_2$ not equivalent; substitution of $ScvO_2$ for $SvO_2$ in calculation of $VO_2$ resulted in unacceptably large errors |
| Kopterides and colleagues [18] | *n* = 37; septic shock | Mean $SvO_2$ below mean $ScvO_2$; mean bias –8.5%<br>95% limits of agreement –20.2 to 3.3%; this resulted in higher $VO_2$ values | $ScvO_2$ and $SvO_2$ not equivalent in ICU patients with septic shock; substitution of $ScvO_2$ for $SvO_2$ in calculation of $VO_2$ resulted in unacceptably large errors |
| Ho and colleagues [19] | *n* = 20; cardiogenic or septic shock | $ScvO_2$ overestimated $SvO_2$ with mean bias 6.9%; 95% limits of agreement –5.0 to 18.8%; changes of $ScvO_2$ and $SvO_2$ did not follow the line of perfect agreement | $ScvO_2$ and $SvO_2$ are not interchangeable numerically |
| van Beest and colleagues [20] | *n* = 53; 265 paired samples; sepsis; ICU; multicentre | Mean $SvO_2$ below mean $ScvO_2$ at all time points; bias of difference 1.7%<br>95% limits of agreement –12.1 to 15.5%; identical results for change in $ScvO_2$ and $SvO_2$<br>Distribution of ($ScvO_2$ – $SvO_2$) (<0 vs. ≥0) similar in survivors and nonsurvivors | $ScvO_2$ does not reliably predict $SvO_2$ in patients with sepsis<br>Trend of $ScvO_2$ not superior in this context<br>$ScvO_2$ – $SvO_2$ ≥0 not associated with improved outcome |
| Scheinmann and colleagues [21] | *n* = 24; critically ill cardiac patients; CCU | $ScvO_2$ levels in superior vena cava are greater than $SvO_2$ in shock (58 ± 13 vs. 47.5 ± 15; *r* = 0.55); changes in $ScvO_2$ reflect changes in $SvO_2$ (*r* = 0.90); $ScvO_2$ from right atrium is similar to $SvO_2$ (49.2 ± 19 vs. 49.2 ± 19; *r* = 0.96) | $SvO_2$ consistently lower than $ScvO_2$<br>Poor correlation in heart failure or shock<br>Changes in $ScvO_2$ reflect changes in $SvO_2$ |
| Dueck and colleagues [25] | *n* = 70; 502 comparative sets; neurosurgery | 95% limits of agreement ranged from 6.8% to 9.3% for single values<br>Correlations between changes of $SvO_2$ and $ScvO_2$: *r* = 0.755, *P* <0.001 | Numerical $ScvO_2$ values not equivalent to $SvO_2$ in varying haemodynamic conditions; trend of $ScvO_2$ may be substituted for the trend of $SvO_2$ |
| Reinhart and colleagues [26] | *n* = 32; critically ill patients; ICU; continuous parallel measurements | $ScvO_2$ closely paralleled $SvO_2$, *in vitro r* = 0.88 and *in vivo r* = 0.81<br>$ScvO_2$ averaged (± SD) 7 ± 4% higher than $SvO_2$<br>$ScvO_2$ changed in parallel in 90% when $SvO_2$ changed more than 5% | Continuous fibreoptic measurement of $ScvO_2$<br>Potentially reliable tool to rapidly warn of acute change in the oxygen supply/demand ratio |
| Ladakis and colleagues [28] | *n* = 31 surgical and *n* = 30 medical; critically ill patients; ICU | Significant difference between mean $ScvO_2$ and $SvO_2$ (69.4 ± 1.1 vs. 68.6 ± 1.2%); *r* = 0.945 for total population | $ScvO_2$ and $SvO_2$ are closely related and interchangeable for initial evaluation |
| Tahvanainen and colleagues [29] | *n* = 42; critically ill patients; ICU; $ScvO_2$ as representative of real changes in pulmonary shunt | Significant correlation between measured variables between PA blood samples and both superior vena cava and right atrial blood samples (*P* <0.001) | $ScvO_2$ can replace $SvO_2$; exact $SvO_2$ value can only be measured from the PA itself |

CCU, cardiac care unit; CI, cardiac index; $DO_2$, oxygen delivery; PA, pulmonary artery; $ScvO_2$, central venous oxygen saturation; $SvO_2$, mixed venous oxygen saturation; $VO_2$, oxygen consumption.

Table 2). $SvO_2$ was particularly reduced in patients with cardiogenic shock or left ventricular failure. Patients with cardiac failure are unable to increase CO during periods of increased oxygen need. Changes in oxygen demand will therefore only be compensated by changes in oxygen extraction in the same direction and indicated by inverse changes in venous oxygen saturations. Consequently, a drop in venous oxygen saturations will be a marker of

**Table 2. Studies describing central venous oxygen saturation in clinical settings**

| Study | Design and subjects | Results | Conclusions |
|---|---|---|---|
| Rady and colleagues [1] | *n* = 36; critically ill patients; ED | Additional therapy is needed after haemodynamic stabilisation to normal blood pressure and heart rate | $ScvO_2$ can be utilised to guide therapy in this phase |
| Pope and colleagues [13] | *n* = 619 registries treated with EGDT; observational study | Groups: $ScvO_2$ <70%, $ScvO_2$ 71 to 89%, $ScvO_2$ >90%<br>Multivariate analysis: initial high $ScvO_2$ higher mortality | Also high $ScvO_2$ values predictive for mortality |
| Ander and colleagues [35] | Controls *n* = 17, high lactate group *n* = 22, low lactate group *n* = 5; chronic congestive heart failure; ED | $ScvO_2$ lower in high lactate group than in low lactate group (32 ± 12% vs. 51 ± 13%) and control (60 ± 6%); after treatment<br>There was a significant decrease of lactate and increase in $ScvO_2$ in the high lactate group compared with the low lactate group | Once patients with decompensated end-stage congestive heart failure are identified, these patients require aggressive alternative management |
| Scalea and colleagues [40] | *n* = 26, trauma patients with suggested blood loss | Despite stable vital signs, 39% of the patients had $ScvO_2$ <65%; these patients required more transfusions; linear regression analysis demonstrated superiority of $ScvO_2$ to predict blood loss compared with normally allowed parameters | $ScvO_2$ is a reliable and sensitive method for detecting blood loss; it is a useful tool in the evaluation of acutely injured patients |
| Di Filippo and colleagues [41] | *n* = 121 brain injury after trauma; noncontrolled study | Nonsurvivors showed higher lactate, lower $ScvO_2$ values; patients with $ScvO_2$ ≤65% showed higher 28-day mortality, ICU LOS and hospital LOS than patients with $ScvO_2$ >65% | $ScvO_2$ <65% in first 24 hours after admission in patients with major trauma and head injury is associated with prolonged hospitalisation and higher mortality |
| Pearse and colleagues [65] | *n* = 118, major surgery | After multivariate analysis, lowest CI and lowest $ScvO_2$ were associated with postoperative complications; optimal $ScvO_2$ cut-off value for morbidity prediction was 64.4%; in the first hour after surgery, significant reductions in $ScvO_2$ were observed, without significant changes in CI or oxygen delivery index | Results suggest that oxygen consumption is also an important determinant of $ScvO_2$; reductions in $ScvO_2$ are independently associated with postoperative complications |
| Rivers and colleagues [73] | *n* = 263; RCT; EGDT vs. controls; severe sepsis, septic shock; ED | EGDT (goal: $ScvO_2$ ≥70%) showed better survival (absolute 16%), lower lactate; more fluids, red cell transfusion and inotropics | EGDT provides benefits to outcome |
| Trzeciak and colleagues [74] | *n* = 16 pre-EGDT; *n* = 22 EGDT | Less PAC utilisation; more fluids and dobutamine used; similar costs | EGDT endpoint can reliably be achieved |
| Kortgen and colleagues [75] | *n* = 30 controls; *n* = 30 septic shock<br>Implementation procedure: septic shock | Implementation: use of dobutamine, insulin, hydrocortisone and activated protein C increased<br>Amount of fluids and packed blood cells unaffected<br>Mortality significantly lower after implementation (27% vs. 53%; $P < 0.05$). | Implementation of sepsis bundle feasible<br>Survival benefit |
| Jones and colleagues [76] | *n* = 79 pre-intervention; *n* = 77 post-intervention; ED | Controls: more renal failure at baseline<br>Greater crystalloid volume and vasopressor infusion<br>Mortality 18 vs. 27% | Implementation resulted in mortality reduction |
| Micek and colleagues [78] | *n* = 60 before implementation order set; *n* = 60 after implementation order set; ED | More appropriate antimicrobial regimen<br>More fluids, more vasopressors<br>Less vasopressor by time of transfer to the ICU | Shorter hospital LOS<br>Lower 28-day mortality |
| Shapiro and colleagues [80] | *n* = 51 historical controls; *n* = 79 septic shock | Patients received more fluids, earlier antibiotics, more vasopressors, tighter glucose control, more frequent assessment of adrenal function, not more packed blood cells | Implementation sepsis protocol feasible<br>No survival benefit |
| Jones and colleagues [94] | Multicentre, randomised; *n* = 300 severe sepsis, septic shock<br>Goals: lactate clearance vs. $ScvO_2$ | Higher in hospital mortality $ScvO_2$; nonsignificant difference (predefined –10% threshold) | No significantly different in-hospital mortality between normalisation of lactate clearance compared with normalisation $ScvO_2$ |

CI, cardiac index; ED, emergency department; EDGT, early goal-directed therapy; LOS, length of stay; PAC, pulmonary artery catheter; RCT, randomised controlled trial; $ScvO_2$, central venous oxygen saturation.

cardiac deterioration. Patients with low venous oxygen saturations in the early disease stage may be considered in shock [34,35]. Also, patients with sepsis and known decreased left ventricular function seem to benefit from early goal-directed therapy (EGDT) when treated for sepsis [36] according to the Surviving Sepsis Campaign guidelines [37]. Finally, in the setting of cardiopulmonary resuscitation, a $ScvO_2$ of 72% is highly predictive for return of spontaneous circulation [38].

#### Trauma

In the initial assessment of trauma patients, an adequate judgement of possible blood loss is essential. Compared with conventional parameters, venous oxygen saturations are superior in predicting blood loss [39,40]. Moreover, after major trauma with brain injury, $ScvO_2$ values below 65% in the first 24 hours are associated with higher mortality (28-day mortality 31.3% vs. 13.5%) and prolonged hospitalisation (45 days vs. 33 days) [41].

#### High-risk surgery

In cardiac surgery patients, $SvO_2$ has been shown to be superior to the mean arterial pressure and heart rate as a qualitative warning sign of substantial haemodynamic deterioration. However, results on the predictive value of $SvO_2$ for CO in clinical settings are inconsistent [42-44]. Nevertheless, continuous $SvO_2$ monitoring enables the early diagnosis of occult bleeding or could show poor tolerance of a moderate anaemia due to the inability of the patient with chronic heart dysfunction or preoperative negative inotropic treatment (for example, β-blockers) to increase CO in the face of anaemia. Furthermore, temporary decreases of $SvO_2$ values after cardiac surgery are of prognostic value and may predict the development of arrhythmias [45-47]. Also, probably due to an increased oxygen extraction ratio, decreased $SvO_2$ values during weaning from mechanical ventilation are predictive for extubation failure [48-50]. Finally, good predictive values of $SvO_2$ for mortality have been described [51,52]. This suggests beneficial effects of $SvO_2$ monitoring, at least during and after cardiac surgery.

Goal-directed therapy has been shown to improve outcome after major general surgery [53]. Originally, the goals in the protocol group were supranormal haemodynamic and oxygen transport values (cardiac index >4.5 l/minute/$m^2$, $DO_2$ >600 ml/minute/$m^2$, $VO_2$ >170 ml/minute/$m^2$). In this group a significant reduction of complications, hospital stay, duration of mechanical ventilation and mortality was achieved when the pulmonary artery catheter was placed preoperatively [54]. Such a strict predefined concept holds certain risks, however, and should not be translated to all patients [55-57]. Meta-analyses of haemodynamic optimisation in high-risk patients revealed haemodynamic optimisation to be beneficial only when interventions were commenced before development of organ failure [58,59]. Several of the studies described showed improved outcome, possibly including long-term survival, when goal-directed therapy was commenced before surgery [54,60-62]. Perhaps owing to methodological shortcomings, a multicentre trial that randomised surgical patients to pulmonary artery catheter guided or conventional management failed to show a difference in outcome [63,64]. More recently a reduction in postoperative complications and duration of hospital stay was described when goal-directed therapy was used postoperatively [65-67]. However, the abovementioned findings do not provide definite answers on how to use venous saturations as a therapeutic goal. Only one interventional trial used $ScvO_2$ as a therapeutic goal in perioperative care [68]. In this study the intervention group received therapy to achieve an estimated oxygen extraction ratio <27% after predefined goals for arterial pressure, urine output, and central venous pressure had been achieved. Fewer patients developed organ failure in the $ScvO_2$ group [68].

#### Sepsis and septic shock

In a large multicentre study, three different cohorts of a very heterogeneous population of critically ill patients were compared for survival after different strategies for haemodynamic therapy had been applied: control versus supranormal values for the cardiac index (>4.5 l/minute/$m^2$) or normal values for mixed venous saturation. In total, the anticipated goal was only achieved in one-third of the patients. There was no significant reduction in morbidity or mortality in any group [69]. An important reason for this may be the late timing of the intervention (that is, after occurrence of organ failure), implying that all patients suffered severe damage and received significant treatment before inclusion.

Global tissue hypoxia as a result of systemic inflammatory response or circulatory failure is an important indicator of shock preceding multiple organ dysfunction syndrome. The development of multiple organ dysfunction syndrome predicts the outcome of the septic patient [37]. Treatment strategies aimed at restoring the balance between $DO_2$ and $VO_2$ by maximising $DO_2$ have not been successful [57,69,70].

In line with studies over several decades [1,21,27, 35,40,71] and based on recommendations [72], Rivers and colleagues randomised 263 patients with severe sepsis or septic shock to standard therapy or EGDT. Compared with the conventionally treated group, the $ScvO_2$ guided group received more fluids, more frequently dobutamine, and more blood transfusion during the first 6 hours. This resulted in an absolute reduction in 28-day mortality of 16% [73].

A large number of studies that implemented certain treatment protocols in the emergency department – including antibiotic therapy and tight glucose control, for example [74-79] – showed a significant decrease in mortality. EGDT endpoints (central venous pressure 8 to 12 mmHg, mean arterial pressure ≥65 mmHg, and $ScvO_2$ ≥70%) can well be achieved in an emergency department setting, suggesting that a multifactor approach is a useful strategy in the treatment of sepsis [74-80]. Of note, three of these studies described similar populations with a high percentage of end-stage renal disease in the control group being prone for higher mortality [76,77,79,81]. Although attainment of $ScvO_2$ >70% has been reported as a prominent factor for survival [82], several studies that used EGDT without this specific target were also able to achieve a survival benefit [83-85]. In summary, as shown by Nguyen and colleagues [86], the use of (modified) EGDT implies early recognition of the critically ill patient and enforces continuous reassessment of treatment. This observation seems to be the greatest gain in the treatment of patients with severe sepsis or septic shock over the past decade.

Earlier studies that enrolled patients admitted to the ICU were unable to show a decrease in mortality after aggressive haemodynamic optimisation [57,69]. In contrast, more recent studies that used modified EGDT protocols were able to show a significant decrease in mortality [85,87,88], suggesting that compliance to dedicated sepsis bundles after the emergency department stage can still be useful.

Low incidences of low $ScvO_2$ values at ICU admission [89] or emergency department presentation [90] do occur together with baseline mortality, however, compared with the original EGDT study [73,89,90]. For clinical appreciation of the above-mentioned results, a thorough look into the data is needed. Interestingly, fewer patients were intubated before the first $ScvO_2$ sampling in the EGDT study [73], and this could partially explain the difference of initial $ScvO_2$ values between both studies [73,89]: due to higher $DO_2$ (pre-oxygenation) and lower $VO_2$ (sedation, paralysis; lower work of breathing), $ScvO_2$ may very well improve in response to emergency intubation in the majority of patients [91]. This hypothesis partially explains the differences between populations [73,89,90] and provides another piece in the puzzle on the value of $ScvO_2$ [92]. Nevertheless, applicability of the results of the EGDT trial may be dependent on the geographical setting and the underlying healthcare system [92,93].

Additionally, no difference in outcome was found between a resuscitation protocol based on lactate clearance and a $ScvO_2$-based protocol [94], and $ScvO_2$ optimisation does not always exclude a decrease in lactate levels [95]. Also, the pursuit of $ScvO_2$ >70% does not always seem to be the optimal solution. Recent data suggest that patients with initially high $ScvO_2$ values may also have adverse outcomes [12,13], probably due to impaired oxygen utilisation. High $ScvO_2$ values may thus represent an inability of the cells to extract oxygen or microcirculatory shunting in sepsis [96].

Finally, as a reflection of an increased respiratory muscle oxygen extraction ratio, a reduced $ScvO_2$ or $SvO_2$ predicts extubation failure in difficult-to-wean patients [48,97]. However, a successful intervention to increase $ScvO_2$ in this context is not yet known. Nevertheless, it is conceivable that in the future $ScvO_2$ will be used as a parameter in weaning protocols for a subset of patients [97,98].

## Continuous measurement

Should continuous measurement be considered when venous saturations are used as a therapeutic goal? It may be argued that changes in venous saturations may occur rapidly, particularly in haemodynamically instable patients, and that discontinuous spot measurements by drawing intermittent blood samples may miss these changes. Accordingly, continuous measurement of $SvO_2$ in septic shock patients revealed a higher frequency of short-term changes in $SvO_2$ in nonsurvivors. Variations in $SvO_2$ could thus be of prognostic importance [99]. However, the lack of therapeutic guidelines and cost-effectiveness issues question the clinical use of continuous measurement of $SvO_2$ in critically ill patients [5,7,58]. Continuous measurement in perioperative care allows detection of fluctuations. Low $SvO_2$ values have been associated with increased complications and morbidity, especially in cardiac surgery [100]. The use of $SvO_2$ values >70% as a target seems promising in cardiac surgery and during cardiopulmonary resuscitation [38,43].

There are currently two commercially available devices to measure $ScvO_2$ continuously. Continuous $ScvO_2$ measurement as part of treatment protocol has shown to be a valuable strategy in the emergency department [71,73] and in cardiac surgery [101]. Additionally, Reinhart and colleagues concluded that continuous $ScvO_2$ measurement in the ICU setting is potentially reliable [26]. However, continuous and intermittent measurements of $SvO_2$ or $ScvO_2$ have never been compared systematically.

## Conclusions

The ongoing debate on differences between $SvO_2$ and $ScvO_2$ and their interchangeability should focus on well-defined populations. $SvO_2$ and $ScvO_2$ are clinically useful but both variables should be used with knowledge and caution. Evaluating the available evidence in a clinical setting, we conclude that low venous oxygen saturations are an important warning sign for the inadequacy of $DO_2$

to meet oxygen demands. Low values may warn the clinician about cardiocirculatory or metabolic impairment and should urge for further diagnostics and appropriate action, whereas normal or high values do not rule out persistent tissue hypoxia. Based on the numerous clues for its usefulness discussed in this article, the use of venous oxygen saturations seems especially useful in the early phase of disease or injury. In clinical practice, venous oxygen saturations should always be used in combination with vital signs and other relevant endpoints.

**Abbreviations**

CO, cardiac output; $DO_2$, systemic oxygen delivery; EGDT, early goal-directed therapy; Hb, haemoglobin; $SaO_2$, arterial oxygen saturation; $ScvO_2$, central venous oxygen saturation; $SvO_2$, mixed venous oxygen saturation; $VO_2$, systemic oxygen demand.

**Competing interests**

The authors declare that they have no competing interests.

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